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CASE REPORT

A Massive Growth Hormone-Secreting Macro-Adenoma of the Pituitary Gland with a Suprasellar Extension: Case Report

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Abstract

Growth hormone-secreting pituitary adenomas also known as somatotropic adenomas are benign tumors arising from somatotroph cells responsible for Growth Hormone (GH) production. Their main clinical manifestation is acromegaly. We report the case of a male patient with a giant pituitary macroadenoma revealed by clinical features of acromegaly and confirmed by biological evidence of GH hypersecretion. The distinctive feature of this case is the exceptional tumor size and its unusual suprasellar extension in contrast to the more common infrasellar growth pattern. This rare presentation highlights the importance of recognizing atypical growth patterns of somatotropic adenomas as they may complicate surgical access and influence the choice of operative strategy. Preoperative MRI plays a pivotal role in accurately characterizing tumor extension and guiding neurosurgical planning.

Key Words: *Macroadenoma; Acromegaly; Growth Hormone (GH); MRI; Invasion; Suprasellar*

Introduction

Pituitary adenomas are benign intracranial tumors and represent one of the most common lesions of the sellar region [1-3]. Although their true prevalence is likely underestimated, they constitute the third most frequent type of brain tumor, with an incidence ranging from 73 to 94 cases per 100,000 individuals [2].

The clinical manifestations of pituitary adenomas typically result from either mass effect or hormone hypersecretion. These can include visual disturbances, headaches, menstrual irregularities, cushing's syndrome and acromegaly [2,4].

Acromegaly is a rare, frequently underdiagnosed disorder caused by chronic overproduction of growth hormone (GH) and insulin-like growth factor 1 (IGF-1) [5-7]. In over 95% of cases, acromegaly is associated with a GH-secreting pituitary adenoma (somatotropinoma) arising from somatotroph cells [5-7].

The annual incidence of acromegaly is estimated at 3 to 5 new cases per million, with a prevalence of approximately 40 to 70 cases

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per million [5,6]. Due to its insidious onset and slow progression, diagnosis is often delayed, typically occurring 4 to over 10 years after the initial symptoms. The average age at diagnosis ranges from 40 to 50 years, although individuals under 20 years old may account for up to 5% of cases. Both sexes are affected equally [1,6,7].

Case Report

A 45-year-old man, with no notable medical history, presented with a one-and-a-half-year history of helmet-like headaches, associated with episodes of vomiting and progressive visual acuity loss. He also reported symptoms suggestive of sleep apnea syndrome.

Clinical examination revealed characteristic features of acromegaly, including acral dysmorphism: enlarged hands and feet, broad and thickened fingers and hypertrophy of soft tissues. Facial abnormalities included a broadened and thickened nose, prominent cheekbones, frontal bossing, thick lips and accentuated facial lines. The remainder of the physical examination was unremarkable (Figures A1 and A2).

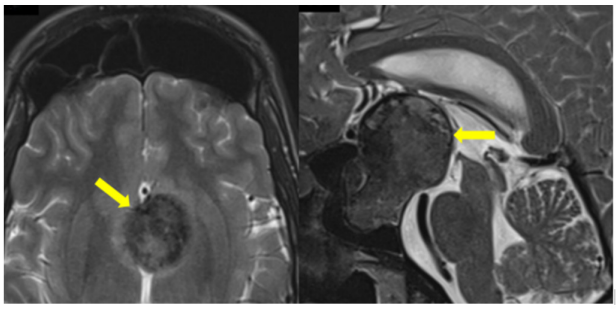
[A1] [A2]

Figure A1 and A2) *Clinical images of the patient's face and profile, illustrating the typical dysmorphic syndrome of acromegaly.*

Biologically, the Growth Hormone (GH) level was markedly elevated at 280.6 ng/mL (normal < 5 ng/mL) and Insulin-like Growth Factor 1 (IGF-1) was also significantly increased to 868.3 ng/mL (normal: 94–284 ng/mL). Follicle-Stimulating Hormone (FSH) and Luteinizing Hormone (LH) were slightly decreased at 1.28 mIU/mL (normal: 1.5–12.4 mIU/mL) and 1.08 mIU/mL (normal: 1.7–8.6 mIU/mL), respectively. C1-inhibitor function was normal. Thyroid function tests revealed a mildly elevated ultrasensitive Thyroid-Stimulating Hormone (TSHus) at 4.42 UI/mL (normal: 0.27–4.2 UI/mL), with slightly decreased levels of Triiodothyronine (T3) at 2.17 pmol/L (normal: 3.1–6.8 pmol/L) and free Thyroxine (T4) at 4.16 ng/L (normal: 7–14 ng/L).

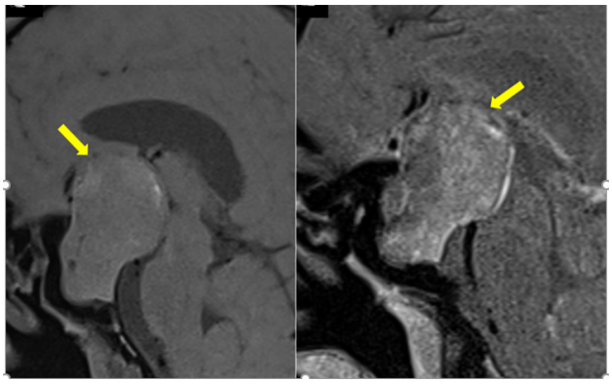
A cranial CT scan revealed a spontaneously hyperdense intra and suprasellar mass, with mild contrast enhancement.

Complementary brain MRI demonstrated an intra-sellar lesion with suprasellar extension, expanding the sella turcica. The lesion appeared isointense on T1-weighted images and heterogeneously hypointense on T2, with hyperintense areas suggestive of necrotic or hemorrhagic changes. Post-gadolinium sequences showed heterogeneous enhancement. The mass measured 52×36×35 mm (height × anteroposterior × transverse) (Figures B1 and B4). Inferiorly, it compressed the sellar floor without invading the sphenoid sinus.



[B1] [B2]

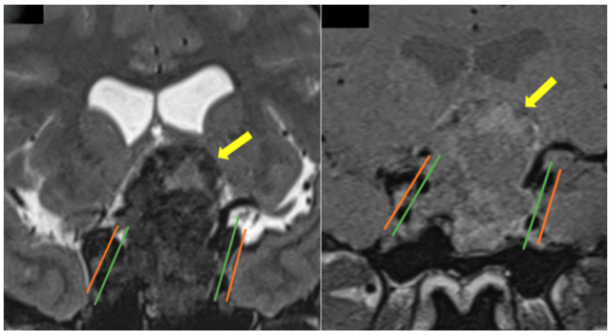
Figure B1 and B2) *Hypothalamic-pituitary MRI (high-resolution). Axial T2-weighted image (B1) and sagittal T2-weighted image (B2) demonstrate a large pituitary macroadenoma with suprasellar extension (yellow arrow). The lesion appears heterogeneous and hypointense on T2, with hyperintense regions at the upper pole suggesting necrotico-hemorrhagic remodeling. Frontal sinus hyperplasia is also visible (B1). High-resolution images allow detailed evaluation of tumor margins and adjacent structures.*



[B3] [B4]

Figure B3 and C4) *Hypothalamic pituitary MRI. T1 weighted images sequences: sagittal pre-contrast image (C1) and sagittal (C2) T1 postcontrast image show a large intensely enhancing sellar and suprasellar mass (yellow arrow).*

Laterally, on the left, it invaded the cavernous sinus, extending between the intercarotid and lateral carotid lines, corresponding to Knosp grade II. On the right, it extended beyond the lateral carotid line at the upper portion of the cavernous sinus, consistent with Knosp grade IIIA (Figures C1 and C2).



[C1] [C2]

Figure C1 and C2) *Hypothalamic-pituitary MRI (high-resolution). Coronal T2-weighted image (B3) and coronal T1 post-contrast image (B4) show bilateral parasellar extension:*

- *Left side: Tumor (yellow arrow) infiltrates the cavernous sinus between the intercarotid line (green) and lateral bi-carotid line (orange), Knosp grade II.*
- *Right side: Tumor (yellow arrow) enters the cavernous sinus crossing the bi-carotid line in the upper cavernous segment, Knosp grade IIIA.*

High-resolution sequences allow clear visualization of the tumor's relationship with the cavernous sinus and internal carotid arteries.

Based on clinical, biological and radiological findings, the diagnosis of acromegaly due to a large GH-secreting pituitary macroadenoma with suprasellar extension and bilateral cavernous sinus invasion (Knosp grade II on the left, grade IIIA on the right) was confirmed.

The patient was scheduled for transsphenoidal surgical resection of the tumor in the following month with planned postoperative follow-up including hormonal assays and control MRI to assess for residual or recurrent disease.

Discussion

Clinical features: Growth Hormone (GH)-secreting pituitary adenomas, also known as somatotropic adenomas, are benign monoclonal tumors arising from the somatotroph cells of the anterior pituitary, which are responsible for GH production [6]. These adenomas can be pure (approximately 60% of cases) or mixed, with some pure somatotropic adenomas co-expressing a free alpha subunit common to glycoprotein hormones such as Follicle-Stimulating Hormone (FSH), Luteinizing Hormone (LH), Thyroid-Stimulating Hormone (TSH) and Human Chorionic Gonadotropin (HCG), sometimes within the same secretory granules as GH [6].

The pathogenesis of somatotropic adenomas remains incompletely understood, with ongoing debate regarding their pituitary versus hypothalamic origin [6]. Somatotroph cells are clearly altered, but the molecular mechanisms leading to clonal expansion are likely multifactorial. Tumorigenesis may involve oncogene activation, while sustained tumor growth could be supported by Growth Hormone-Releasing Hormone (GHRH) or other growth factors such as basic Fibroblast Growth Factor (bFGF) [6].

Clinically, these adenomas most frequently present as acromegaly [6]. The symptomatology results from both local mass effects and systemic consequences of chronic GH and

IGF-1 hypersecretion, potentially leading to significant comorbidities and impaired quality of life [6-9]. The most characteristic clinical features include:

- **Acral dysmorphism**: Enlargement of hands and feet due to soft tissue hypertrophy, causing increased shoe, glove and ring sizes. Fingers are broadened and thickened, often described as "spade-like"-which matched our patient's presentation [6,7].
- **Orofacial changes**: Frontal bossing, widened nasal bridge, enlarged nose and ears, thick lips, prominent nasolabial folds, coarse facial features and mandibular prognathism leading to dental malocclusion. In our patient, imaging revealed cranial vault thickening, hypertrophy of frontal sinuses and mandibular angle widening (Figure B1) [1,6,7].
- **Headache**: Reported in ~60% of cases, sometimes severe and disproportionate to tumor size.
- **Visual disturbances**: Due to upward tumor growth compressing the optic chiasm, as observed in our patient [6-8].

Other systemic manifestations include macroglossia contributing to obstructive sleep apnea, deepening of the voice (from laryngeal and sinus hypertrophy), arthralgia, menstrual irregularities, erectile dysfunction, hyperhidrosis, hypertension, impaired glucose tolerance or diabetes and progressive cardiopulmonary dysfunction. In pediatric patients, gigantism may occur when epiphyseal fusion is incomplete [6,7].

Diagnosis is first suspected clinically and confirmed by biochemical testing. It is established by demonstrating elevated serum GH levels that are not suppressed during an Oral Glucose Tolerance Test (OGTT), along with increased serum IGF-1 concentrations adjusted for age and sex [6,7].

Due to the often insidious progression of symptoms, diagnosis is typically delayed and many patients present with advanced adenomatous growth at initial evaluation [1,6,7].

Radiological Evaluation

Imaging is crucial not only to confirm the presence of a pituitary adenoma but also to assess the invasion of adjacent structures and guide follow-up [10]. Pituitary macroadenomas are defined as tumors exceeding 1 cm in diameter. Most are non-functioning and chromophobic, whereas somatotropic adenomas are generally smaller on average [1,9].

In this case, the adenoma was exceptionally large, with marked suprasellar and parasellar extension, contrasting with the typical infrasellar growth pattern described in most GH-secreting adenomas, which often invade the sphenoid sinus and clivus due to proteolytic enzyme secretion [9]. Possible explanations for this atypical growth include:

- Delayed diagnosis, allowing prolonged tumor expansion.
- Anatomical variations, such as a more compliant diaphragma sellae, facilitating suprasellar spread.
- Tumor-specific biological factors, including local secretion of growth factors or proteases enhancing expansion [9].

On CT, these tumors typically appear iso to hypodense relative to normal pituitary tissue; hyperdensity may reflect calcifications or hemorrhage, as seen in our patient [1].

MRI is the gold standard for pituitary adenoma evaluation. On T1-weighted sequences, adenomas usually appear hypo to isointense

relative to normal pituitary tissue and white matter structures (e.g., semi-oval centers, brainstem profile) (Figure B3). On T2-weighted sequences, adenomas are iso- to hyperintense, with variable contrast enhancement (Figures B4 and C2). Areas of intratumoral necrosis appear hypointense on T1 and hyperintense on T2, while hemorrhagic components are hyperintense on both T1 and T2 (Figures B1-B3 and C1) [1,6]. MRI allows precise assessment of tumor size, location, invasiveness and internal characteristics (necrotic, cystic, or hemorrhagic) [1,3,4,6,10-12].

Cavernous Sinus Invasion

Cavernous Sinus Invasion (CSI) is associated with incomplete surgical resection, persistent hormonal abnormalities and technical challenges during surgery [4,10,11,13-15].

The Knosp grading system, integrated into the 2017 WHO pituitary tumor classification, provides an MRI-based, standardized assessment of parasellar extension [14]. Additional radiological parameters, including Percentage of Encasement of the Internal Carotid Artery (PEICA), Venous Compartment Obliteration (VCO) and the Fernandez-Miranda anatomical classification, enhance preoperative detection of CSI [3,4,11,14]. Proton-Density-Weighted Imaging (PDWI) has also been highlighted for its high sensitivity in detecting CSI and guiding safe, complete tumor resection [3].

T2-hyperintense somatotropinomas have been shown to carry a significantly higher risk of CSI compared to T2-hypointense tumors. In a study of 210 GH-secreting macroadenomas larger than 11 mm, CSI was found in 56% of T2-hyperintense tumors versus 28% of T2-hypointense tumors [10].

Therapeutic Management

Treatment goals in acromegaly include controlling hormonal hypersecretion, reducing tumor volume, relieving symptoms and preventing recurrence thereby improving long-term morbidity and mortality [6].

Transsphenoidal surgery remains the first-line treatment and is the most effective method for rapidly normalizing GH and IGF-1 levels [6]. When surgery is contraindicated or unsuccessful, medical therapy (somatostatin analogs, GH antagonists) or radiotherapy may be indicated.

Despite biochemical remission, many patients continue to experience residual joint pain, deformities and reduced quality of life due to irreversible tissue changes [6].

Novelty and Clinical Implications

This case highlights the rarity of a GH-secreting macroadenoma with suprasellar extension, scarcely documented in the literature. Its recognition is critical for anticipating surgical challenges, adapting operative strategies and informing prognosis. The observation underscores the importance of MRI in accurately delineating tumor extension and guiding individualized therapeutic approaches.

Conclusion

Invasive somatotropic pituitary adenoma, the underlying cause of secondary acromegaly is a rare but serious condition associated with significant morbidity and mortality. A thorough understanding of its pathophysiology, as well as its clinical, biochemical and radiological features are essential to ensure optimal management and improve the prognosis of affected patients.

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